

Expert Opinion

Functional/Psychogenic Neurological Symptoms and Headache

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Key words: functional/psychogenic neurological symptom, headache attributed to somatization disorder, conversion disorder

(*Headache* 2011;51:781-788)

“If we could only show that the problem is in the brain then they wouldn’t think it was all in their heads!” (Senior Doctor at a meeting on chronic fatigue syndrome, NIH)

Functional (or psychogenic) neurological symptoms are common in neurology. Around one-third of neurology outpatients have symptoms that neurologists rate as only “somewhat” or “not at all” explained by disease. A recent study of 3781 new neurology outpatients in Scotland found that around 5% had a primary diagnosis of a functional/psychogenic/conversion symptom such as non-epileptic attacks or functional weakness.¹ Many studies have shown that these symptoms often persist, are associated with distress and disability²⁻⁵ and a low rate of misdiagnosis.^{1,6}

Despite their frequency in neurological practice, neurologists still have an uneasy relationship with this area of their practice. Although most neurologists recognize the phenomenon of functional/psychogenic symptoms, many try to ignore it either by making no diagnosis at all,⁷ or by regarding it as a psychiatric disorder which it is not their responsibility to manage.⁸

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In a recent survey of neurologists,⁹ “psychogenic/functional disorder” very clearly came bottom of a list of “most likeable neurological conditions to treat” – even low back pain was more popular.

Neurologists with an interest in headache have, in my own experience, particular antibodies to admitting to a relationship of functional/psychogenic symptoms with headache disorders. This comes, I believe, firstly from an understandable desire to “stick up” for headache patients who have often had trouble being taken seriously, but also from a quite widely held view by many neurologists that patients with functional/psychogenic neurological symptoms, in distinction to headache patients, commonly feign their symptoms.¹⁰

So, I am aware that I am stepping into a lion’s den in accepting Dr. Evans’ invitation to comment on these 2 cases describing an intersection between psychogenic/functional symptoms and headache. But perhaps I can persuade you that there is an intriguing relationship between headache and functional/psychogenic symptoms that is relevant to many patients seen by headache specialists and one which deserves further impartial study.

The term functional symptoms (rather than psychogenic/non-organic/conversion, etc) will be used in this article. The rationale will be discussed in the text. To assist the discussion, I will present the results of a small survey of headache experts carried out with Dr. Evans in relation to these topics (Appendix I).

CLINICAL HISTORIES

Case 1.—This 41-year-old right-handed woman was seen in urgent neurological consultation with a chief complaint of headache and left-sided paralysis. She had just completed an upper endoscopy as an outpatient with findings of gastritis and was in recovery when she complained of a bitemporal and behind-the-eyes throbbing headache with an intensity of 10/10 associated with nausea, light and noise sensitivity. At the onset of the headache, she reported that she could not at all move the left side of her body and had tingling of the left side as well.

There was a prior history of exactly similar headaches since the age of 15 still occurring about 3 times per week for years without aura for which she would take an opioid combination analgesic and the headache would last up to 2 days. She had been on topiramate 100 mg daily for prevention with a mild reduction in frequency and was followed by her primary care physician.

She reported a history of similar episodes of paralysis of either the left or right side associated with similar headaches since the age of 20, the last about 3 weeks prior affecting the left side lasting about 2 days, the previous in 2003. The episodes of paresis have lasted up to 1 week. She had seen a neurologist in the past and reported that the episodes were due to migraine, but medical records were not available.

There was a past medical history of hypertension on lisinopril. There was no family history of migraine or stroke.

Vital signs were normal. She was not emotionally distressed. Neurological examination revealed initially slurred speech which became clear with encouragement but no aphasia. Cranial nerves were intact. There was a normal facial sensory exam and motor function. Motor exam was normal except for the left upper and lower extremity which was initially 0/5 proximally and distally. I held her left arm over her face and advised her that I was going to “lock” it into place to further evaluate her condition. The left arm remained suspended in the air for a minute. Hoover’s test was positive on the left (I placed my right hand under the left heel; when the normal right hip was flexed against resistance, I could feel the left heel pushing down). I then encouraged the patient and

told her that if we exercised the left side, perhaps we might get some strength back. With encouragement, the left upper extremity was at least 4/5 proximally and distally and the left lower extremity 1-2/5. Sensory exam was normal on the right, decreased on the left. Deep tendon reflexes were 1+ and symmetric. Plantars were flexor. Gait was not initially tested.

An initial computed tomography of the brain was negative. The next day, the headache had resolved after treatment with acetaminophen with codeine. On exam, the right upper extremity was 4/5, the right lower extremity 0/5. A magnetic resonance imaging of the brain was normal. She could stand with assistance only but stated that she could not walk. She was seen by a physical therapist. A psychiatry consultant reported evidence of an anxiety disorder. By the next day, she was walking using a tray table with a dragging monoplegic gait.

Case 2.—This is a 42-year-old woman seen in headache consultation with daily constant headaches from the onset for 5 months described as a generalized constant pressure with an intensity of 2-10/10 with an average of 4/10 without nausea, light or noise sensitivity, or visual symptoms. She occasionally would take ibuprofen, which might dull the pain. Tizanidine briefly helped but hydrocodone did not help.

She was given intravenous dihydroergotamine initially, which did not help while intravenous chlorpromazine helped temporarily. Topiramate, nadolol, and indomethacin have not helped reduce the frequency of headaches.

There was a prior history of headaches for about 20 years occurring about 2-3 times per year. She could not recall the location of the prior headaches or whether she had associated nausea, an aura or triggers but did recall light and noise sensitivity. She stated that rizatriptan would provide rapid relief.

She reported 2 passing out spells, 5 months and 10 days ago, where she was observed to be shaking without tongue biting or incontinence and perhaps confusion following. She has had complaints of intermittent stuttering and shaking of the hands. She was evaluated by a neurologist who diagnosed psychogenic stuttering and a non-physiological tremor. A magnetic resonance imaging of the brain was normal. A routine electroencephalogram (EEG) was normal.

She had a 24-hour video EEG 1 week ago reported as showing 3 typical spells with closing her eyes, clenching her jaws, with limp arms and legs, sometimes shaking lasting for 5-10 seconds but with a normal EEG.

There was no history of anxiety or depression. However, she reported being under lots of stress with a back injury, loss of her job, and the illness of her father. She has not seen a psychologist or psychiatrist.

The past medical history was otherwise negative. Her mother and sister have migraines. Neurological examination was normal.

On discussion, she told me that she had been diagnosed with pseudoseizures but assured me that she was not crazy and wanted to know what was really wrong. After discussion of the role of stress in triggering symptoms, I placed her on venlafaxine and recommended a psychiatry consultation.

Questions.—What is the relationship between headache and functional/psychogenic symptoms? How might you distinguish functional/psychogenic weakness from hemiplegic migraine? Does psychogenic headache exist? How might you diagnose it? What language is best to explain these problems to the patient? What treatment would you recommend?

EXPERT OPINION

Review of Cases.—Case 1 describes a patient with a clear-cut history of migraine who develops a hemiparesis in association with an apparent recurrence of migraine while coming round from an endoscopy.

The hemiparesis is clearly identified as functional on the basis of several positive signs: she has a positive Hoover's sign, the weak arm remained temporarily suspended in mid air before collapsing and her weakness improved with simple encouragement. The next day she had a dragging monoplegic gait seen typically in functional hemiparesis.¹¹ What started out as migraine (possibly with hemisensory and hemiparetic aura) has ended up as functional hemiparesis.

The history of prior episodes suggests either a prior history of migraine with aura or perhaps previous similar episodes of functional weakness in association with migraine.

A further important factor here is the setting in which this took place. The patient was recovering from an endoscopy and no doubt had sedation. Anesthetics are a recognized trigger for functional symptoms, especially dissociative (non-epileptic) attacks.^{12,13} The helpless bustle of the recovery room, combined with the “spaced out” experience of benzodiazepines, is a fertile environment for dissociative experiences and functional symptoms.¹⁴

Case 2 describes a patient with “New Daily Persistent Headache” (International Headache Society [IHS]-2, 4.8) on a background of probable migraine. In the context of this headache, the patient has developed clear evidence of dissociative (non-epileptic) attacks with typical features being closed eyes, long attacks, and shaking of flaccid limbs. The normal EEG is helpful, too, but EEG can be normal in frontal lobe seizures and it is really the typical features of the attack that should give the clinician confidence to make the diagnosis. The presence of functional stuttering and a functional tremor also make this diagnosis clearer. In this case there is no relationship between worsening headache and her attacks, but is it a coincidence that she has these 2 physical symptoms along with back pain and is “under stress”?

Migraine and Functional Weakness.—Case 1 is similar to many that I have come across in my own clinical practice. In a case series of 107 patients with functional weakness,¹⁵ 48% had an acute onset (n = 51). Of these, 8 patients developed functional weakness in the context of a migraine, probably with aura. A further two occurred in patients coming out of an anesthetic (data in preparation). Panic symptoms, dissociative symptoms, acute pain, and physical injury were other common associated factors at onset. Headache was a complaint of 40% of patients with functional weakness (n = 107) compared to 9% of neurological weakness controls (n = 46).¹⁵

There are many possible interactions between migraine and functional symptoms including:

1. Migrainous aura somatic symptom as a trigger for the same type of functional neurological symptom – as in Case 1.

2. Migraine inducing an altered state of consciousness or fatigue in which functional neurological symptoms are more likely to spontaneously occur, eg, migrainous fatigue state allowing the emergence of a dissociative (non-epileptic) attack.
3. The presence of migraine in someone who also has functional neurological symptoms. Migraine is associated to some degree with depression and anxiety, which in turn is associated with functional neurological symptoms.
4. The worsening of pre-existing functional neurological symptoms at times of migraine.

In a useful study, Young et al described 24 patients with migraine who presented like Case 1.¹⁶ They describe “unexplained motor symptoms” characterized by “give-way” weakness (ie, functional weakness). The authors¹⁶ and the accompanying editorial¹⁷ were keen to point out that these symptoms were genuine (which I would agree with) but opted to explain them on the basis of “a disordered protective reflex similar to that which causes give-way weakness in an injured limb” rather than see them tarred with a “psychogenic” brush. In this study, the Minnesota Multiphasic Personality Inventory (MMPI) was used to “rule out” psychogenic disorder, even though the MMPI has performed poorly in studies attempting to distinguish patients with functional/psychogenic symptoms from disease controls.¹⁸ Just as in complex regional pain syndrome, where motor symptoms also have clear-cut “functional” characteristics,¹⁹ the curious bias of many physicians is that only biological explanations are suitable in a situation where you “believe the patient.”

A “disordered protective reflex” is certainly a plausible factor, but there is a broader hypothesis. Could it be that a patient, frightened and in pain with migraine, remembering what happened the last time they had paralysis, develops depersonalization symptoms, which are involuntarily amplified by attention and concern paid to the abnormal limb thereby bringing about and perpetuating the symptom? Bringing cognitions and emotions in to the formulation does not do a disservice to the patient; it helps everyone understand it better. Studies of abnormal brain functioning patients with

functional/psychogenic symptoms (in the absence of migraine or pain) are starting to unravel the neural mechanisms of these symptoms in a way that does not exclude psychological formulation but is complementary to it.²⁰⁻²³ Furthermore, there have been reports of functional weakness arising from all categories of neurological disease and not just migraine.²⁴⁻²⁷

The prior literature on a possible relationship between psychogenic symptoms and migraine is sparse. Babinski published a series of 4 patients with “migraine ophthalmique hystérique” who had convulsions and persistent unilateral symptoms in association with migraine.²⁸ One study described 4 patients with “psychogenic basilar migraine” who had functional symptoms in association with migraine, but they concluded that attacks were all psychogenic in nature and did not suggest a relationship between the two.²⁹ Another study reported psychogenic blindness complicating migraine.³⁰ Finally, a recent study from India reported an astonishingly high rate of swooning dissociative (non-epileptic) attacks in 23% of 656 unselected female adults with migraine, with attacks typically occurring in the context of migraine.³¹ This is clearly at odds with published experience, although a cultural phenomenon cannot be excluded.

The Patient With Functional/Psychogenic Symptoms and New Daily Persistent Headache – Could the Headache Be Psychogenic Too?—Case 2 raises the familiar problem of how to assess headache in the context of a patient with many other somatic symptoms – in this case dissociative (non-epileptic) attacks, tremor, and speech disturbance. We are not told much about her back pain, but it seems likely not to relate to underlying structural disease either. Assuming the absence of a secondary cause, most neurologists would recognize the pattern of her headache as new daily persistent headache (NDPH). About 10% have a preceding stressful life event as in this case.

There are 2 main ways of dealing with this. First, you could simply make a diagnosis of NDPH and advise appropriately. This is straightforward and allows the headache specialist a way of not having to worry too much about all those other messy symptoms the patient presents with.

Alternatively, you could step back and wonder if perhaps her headache is a pain syndrome which is best understood in the context of a generalized vulnerability to somatic symptoms including back pain, dissociative attacks, tremor (and probably fatigue). By association with other “psychogenic” symptoms, does this mean that it is a “psychogenic headache”?

“Psychogenic headache” can be used to mean anything from a headache where psychological factors are of dominant importance to a headache that is reported but which the patient is not actually experiencing. If a patient reports a “20/10” headache pain rating but looks undistressed, is the patient exaggerating, imagining, or just innumerate? The term “psychogenic headache” has been used in so many different ways³² that, in my view, the term is meaningless and has rightly disappeared from classifications.

Perhaps then it is one of the 2 headache disorders attributed to psychiatric disorder in the IHS-2? “Headache attributed to psychotic disorder” (IHS-2, 12.1) describes a headache clearly based on a delusion, for example, that there is a transmitter in the brain – it is not that. The other is “headache attributed to somatization disorder” (IHS-2, 12.2). It may well fit the latter, although for this diagnosis patients must have 4 pain symptoms, 2 gastrointestinal symptoms, and 1 sexual symptom (as well as 1 functional neurological symptom). What if she is 1 symptom short? In addition, the symptomatology of Case 2’s headache is really rather typical of NDPH. “Headache attributed to somatization disorder” as a category does not really resolve the problem posed by this case. First, the clinical features of this headache in this category are not well defined (57% of headache specialists agreed with this in the survey – see Appendix I). Second, it is anomalous with the way other branches of medicine have chosen to deal with their more problematic symptoms. Gastroenterologists, for example, diagnose irritable bowel syndrome. It remains irritable bowel syndrome even when associated with other functional symptoms such as back pain or somatization disorder.

So we come back to a diagnosis of NDPH, which is a mysterious primary disorder of unknown etiology where various hypotheses include an infectious ante-

cedent, defective internal jugular venous drainage, and cervical joint hypermobility. The phenotype in this case is the same as chronic tension type. Might there be a similar neurobiology to chronic tension type headache, which is increasingly associated with a wide range of interesting structural, electromyographic, and neurochemical abnormalities mostly operating at the more distal end of the nervous system?³³ As with migraine, patients can be reassured; they are not crazy, science believes them. Most models of the pathophysiology of chronic tension type headache, however, contain a bidirectional arrow pointing to “central” or “limbic” control centers. Neuroimaging studies of chronic pain have delineated these circuits in more detail, linking these to psychological mechanisms such as attention, emotion, catastrophization, and ideas about illness.³⁴ In Case 2, because of the wide variety of symptoms seen, it seems that these “central” mechanisms, rather than being a secondary effect, may instead be the key to understanding many of the patients’ other symptoms, too, whether or not they have peripheral neurochemical correlates. The IHS struggles to capture this complexity and arguably perhaps it should not try to.

What If Functional Neurological Symptoms Were Considered As Legitimate As Migraine?—For both these cases, the problem may lie in the fact that many neurologists have difficulty taking seriously or believing patients with functional weakness and dissociative attacks. There is still widespread support among neurologists for the idea that deception is playing a part in these symptoms even if they do not think it is the whole story.¹⁰ Certainly, 64% of the respondents of our survey agreed that these patients often deliberately exaggerate their symptoms (see Appendix I). This may be in large part because of the way that functional motor symptoms in particular can be shown at the bedside to be inconsistent (whereas there is no similar test for headache).

No wonder, then, that headache specialists do not want “honest” headache patients to be associated with “dishonest” functional/psychogenic patients. Only 9% of headache specialists thought that patients with headaches exaggerate.

But imagine how much more interesting the debate would be, if both types of symptoms could be

taken equally seriously. In that scenario we could ask much more interesting questions not only about the biology of functional neurological symptoms but about the psychology of headache.

What Should These Patients Be Told About Their Symptoms and the Relationship to Their Headache? What Treatment Should Be Given?—A different attitude to functional neurological symptoms not only has implications for how these symptoms interface with the rest of the neurology, it changes the way you communicate the diagnosis to patients. Successful explanation of the diagnosis can in itself be highly therapeutic. I have outlined an approach elsewhere¹¹ which involves the following steps:

- Explain to the patient what they do have – I personally use “*functional weakness/movement disorder*” and “*dissociative attacks*.” Use psychogenic if you wish but be aware that for most patients, words like “psychosomatic” and “psychogenic” mean “making it up.”³⁵
- Explain why you are making the diagnosis – I show patients their Hoover’s sign or discuss why their attacks can only be dissociative. This gives the diagnosis more authority and logic.
- Explain what they do not have – eg, multiple sclerosis, epilepsy, and why.
- Tell the patient you believe them – eg, “*I don’t think you are making up or imagining these symptoms/going crazy.*”
- Emphasize that it is common – eg, “*I see lots of patients with similar symptoms.*”
- Emphasize the potential for reversibility – eg, “*because the nervous system is not damaged these symptoms have the potential to improve.*”
- Metaphors may be useful – eg, “*this is like a software problem with the brain rather than a hardware problem.*”
- Introducing the role of psychological factors – eg, “*this problem is not ‘all in your mind’ but the way that you think about things can affect it. Look at that Hoover’s sign. When you were thinking hard about moving your weak leg it didn’t work but then when you were distracted by moving your good leg, your weak leg moved normally.*”

- Use written information – eg, information at <http://www.neurosymbols.org> (a self-help website by the author of this article) or <http://www.nonepilepticattacks.info>. I personally always copy my letters to patients as well.
- Involve family and friends – they need to understand what these symptoms are as well.

Explanation can go a very long way in these symptoms. For persistent symptoms physiotherapy, utilizing encouragement, distraction techniques, and graded exercise can be useful. Cognitive behavioral therapy exploring patterns of thinking and behavior over a longer period of time can also be helpful – currently only dissociative attacks have a clear evidence base for this kind of treatment.³⁶ Other forms of psychotherapy may also be useful but need to be conducted by someone who is familiar with functional neurological symptoms. There is little evidence regarding antidepressants in functional neurological symptoms. However, patients with these symptoms frequently have symptoms such as depression, anxiety, pain, or insomnia for which these agents may be considered anyway.

I would explain to Case 1 that she had experienced a migraine with aura and this had triggered functional weakness. I would discuss how sometimes a “pattern” gets set up in the brain so that when the migraine keeps happening then functional weakness keeps getting triggered as well. I would also explore whether the patient is panicking or experiencing dissociative symptoms when she gets migraine with aura as this may be exacerbating the weakness.

I would explain to Case 2 that she had NDPH, which was related to various changes in the nervous system, most prominently an increase in the “volume knob” from the pain-sensitive structures in her head. I would also explain how her functional neurological symptoms commonly occur with other symptoms such as headache, pain, and anxiety, so it might be best to see this as a single general vulnerability to multiple symptoms rather than multiple different problems.

Acknowledgments: Dr. Stone thanks Richard Daventport, Department of Clinical Neurosciences, Western General Hospital, Edinburgh, UK, for helpful comments. We thank the Southern Headache Society respondents for participating in the survey.

APPENDIX I

Email survey of 98 headache experts using the Southern Headache Society (USA) listserv, September 2010. There were 33 respondents, 76% male with a mean of 19 years' experience in practice:

	Strongly Disagree	Disagree	Neither Agree nor Disagree	Agree	Strongly Agree
Patients with headaches often deliberately exaggerate the severity of their headache symptoms	9%	52%	30%	9%	0%
Patients with psychogenic/functional symptoms such as non-epileptic attacks or "non-organic" weakness often deliberately exaggerate the severity of their symptoms	6%	12%	18%	46%	18%
It is possible for a patient to report headache even though they are not experiencing it and not malingering (one definition of "psychogenic headache")*	0%	15%	18%	61%	6%
Headache due to somatization disorder (IHS-2, 12.1)** is a useful diagnostic category	6%	22%	16%	53%	3%
Headache due to somatization disorder (IHS-2, 12.1)** is clinically distinct from other forms of headache	3%	21%	33%	33%	9%

*Frequency of seeing this kind of patient in last 5 years was given as "0" = 16%, "1-5" = 39%, "6-20" = 19%, "over 20" = 26%.

**Definition was provided.

REFERENCES

- Stone J, Carson A, Duncan R, et al. Symptoms "unexplained by organic disease" in 1144 new neurology out-patients: How often does the diagnosis change at follow-up? *Brain*. 2009;132(Pt 10):2878-2888.
- Sharpe M, Stone J, Hibberd C, et al. Neurology out-patients with symptoms unexplained by disease: Illness beliefs and financial benefits predict 1-year outcome. *Psychol Med*. 2010;40:689-698.
- Stone J, Sharpe M, Rothwell PM, Warlow CP. The 12-year prognosis of unilateral functional weakness and sensory disturbance. *J Neurol Neurosurg Psychiatry*. 2003;74:591-596.
- Ibrahim NM, Martino D, van de Warrenburg BP, et al. The prognosis of fixed dystonia: A follow-up study. *Parkinsonism Relat Disord*. 2009;15:592-597.
- Reuber M, Pukrop R, Bauer J, Helmstaedter C, Tessaendorf N, Elger CE. Outcome in psychogenic non-epileptic seizures: 1- to 10-year follow-up in 164 patients. *Ann Neurol*. 2003;53:305-311.
- Stone J, Smyth R, Carson A, et al. Systematic review of misdiagnosis of conversion symptoms and "hysteria." *BMJ*. 2005;331:989.
- Friedman JH, LaFrance WC Jr. Psychogenic disorders: The need to speak plainly. *Arch Neurol*. 2010;67:753-755.
- Espay AJ, Goldenhar LM, Voon V, Schrag A, Burton N, Lang AE. Opinions and clinical practices related to diagnosing and managing patients with psychogenic movement disorders: An international survey of movement disorder society members. *Mov Disord*. 2009;24:1366-1374.
- Evans RW, Evans RE. A survey of neurologists on the likeability of headaches and other neurological disorders. *Headache*. 2010;50:1126-1129.
- Kanaan R, Armstrong D, Barnes P, Wessely S. In the psychiatrist's chair: How neurologists understand conversion disorder. *Brain*. 2009;132(Pt 10):2889-2896.
- Stone J. The bare essentials: Functional symptoms in neurology. *Pract Neurol*. 2009;9:179-189.

12. Lichter I, Goldstein LH, Toone BK, Mellers JD. Nonepileptic seizures following general anesthetics: A report of five cases. *Epilepsy Behav.* 2004;5:1005-1013.
13. Reuber M, Enright SM, Goulding PJ. Postoperative pseudostatus: Not everything that shakes is epilepsy. *Anaesthesia.* 2000;55:74-78.
14. Stone J. Dissociation: What is it and why is it important? *Pract Neurol.* 2006;6:308-313.
15. Stone J, Warlow C, Sharpe M. The symptom of functional weakness: A controlled study of 107 patients. *Brain.* 2010;133:1537-1551.
16. Young WB, Gangal KS, Aponte RJ, Kaiser RS. Migraine with unilateral motor symptoms: A case-control study. *J Neurol Neurosurg Psychiatry.* 2007;78:600-604.
17. Goadsby PJ. MUMS the word. Migraine with unilateral motor symptoms: What can you say? *J Neurol Neurosurg Psychiatry.* 2007;78:553.
18. Kalogjera-Sackellares D, Sackellares JC. Analysis of MMPI patterns in patients with psychogenic pseudoseizures. *Seizure.* 1997;6:419-427.
19. Birklein F, Riedl B, Sieweke N, Weber M, Neundorfer B. Neurological findings in complex regional pain syndromes – analysis of 145 cases. *Acta Neurol Scand.* 2000;101:262-269.
20. Voon V, Brezing C, Gallea C, et al. Emotional stimuli and motor conversion disorder. *Brain.* 2010;133(Pt 5):1526-1536.
21. Cojan Y, Waber L, Carruzzo A, Vuilleumier P. Motor inhibition in hysterical conversion paralysis. *Neuroimage.* 2009;47:1026-1037.
22. Vuilleumier P, Chicherio C, Assal F, Schwartz S, Slosman D, Landis T. Functional neuroanatomical correlates of hysterical sensorimotor loss. *Brain.* 2001;124(Pt 6):1077-1090.
23. Voon V, Gallea C, Hattori N, Bruno M, Ekanayake V, Hallett M. The involuntary nature of conversion disorder. *Neurology.* 2010;74:223-228.
24. Caplan LR, Nadelson T. Multiple sclerosis and hysteria. Lessons learned from their association. *JAMA.* 1980;243:2418-2421.
25. Onofrj M, Bonanni L, Manzoli L, Thomas A. Cohort study on somatoform disorders in Parkinson disease and dementia with Lewy bodies. *Neurology.* 2010;74:1598-1606.
26. Eames P. Hysteria following brain injury. *J Neurol Neurosurg Psychiatry.* 1992;55:1046-1053.
27. Crimlisk HL, Bhatia K, Cope H, David A, Marsden CD, Ron MA. Slater revisited: 6 year follow up study of patients with medically unexplained motor symptoms. *BMJ.* 1998;316:582-586.
28. Babinski J. De la migraine ophthalmique hystérique. *Arch Neurol.* 1890;20:305-335.
29. Sanchez-Villasenor F, Devinsky O, Hainline B, Weinreb H, Luciano D, Vazquez B. Psychogenic basilar migraine: Report of four cases. *Neurology.* 1995;45:1291-1294.
30. Ziegler DK, Schlemmer RB. Familial psychogenic blindness and headache: A case study. *J Clin Psychiatry.* 1994;55:114-117.
31. Chakravarty A, Mukherjee A, Roy D. Migraine, epileptic seizures and psychogenic non-epileptic seizures: Observations in Indian patients in a clinic-based study. *Neurol India.* 2010;58:631-633.
32. Packard RC. What is psychogenic headache? *Headache.* 1976;16:20-23.
33. Fumal A, Schoenen J. Tension-type headache: Current research and clinical management. *Lancet Neurol.* 2008;7:70-83.
34. Wiech K, Kalisch R, Weiskopf N, Pleger B, Stephan KE, Dolan RJ. Anterolateral prefrontal cortex mediates the analgesic effect of expected and perceived control over pain. *J Neurosci.* 2006;26:11501-11509.
35. Stone J, Wojcik W, Durrance D, et al. What should we say to patients with symptoms unexplained by disease? The “number needed to offend.” *BMJ.* 2002;325:1449-1450.
36. Goldstein LH, Chalder T, Chigwedere C, Khondoker MR, Moriarty J, Toone BK, Mellers JD. Cognitive-behavioral therapy for psychogenic nonepileptic seizures: A pilot RCT. *Neurology.* 2010;74:1986-1994.